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URETHRAL FUNCTION FOLLOWING VAGINAL OR CESAREAN DELIVERY.

Aims of Study

Vaginal delivery is implicated as a significant aetiological factor in the development of urinary incontinence. However though the prevalence of urinary incontinence is as high as 35% after delivery ,[1] the pathophysiology is still unclear.

Pelvic floor denervation reported in 80% after vaginal delivery but recovers in 60% at 2 months postpartum.[2] Urethral support defects noted in 50% after vaginal delivery lead to alterations in bladder neck position & ability to contract the pelvic floor.[3] However neither of these studies correlated symptoms with pelvic floor damage. Cystometry in pregnancy and postpartum also does not explain the high prevalence of urinary symptoms.[4]

Pressure-flow studies have been used to assess urethral resistance.[5] Using this method the parameters which can be measured are the detrusor pressure pressure as urinary flow commences urethral opening pressure (UOP), detrusor pressure at the end of voiding, urethral closure pressure (UCP). Women with urodynamic stress incontinence have lower opening and closure pressures than women with competent urethral sphincters.

The aim of our study was to evaluate the effect of childbirth on urethral resistance using pressure flow analysis and to determine the relationship of this pressure flow data to urinary incontinence.

Methods

This was a prospective longitudinal study in a London teaching hospital. All nulliparous women in their first pregnancy were invited to participate. Exclusion criteria included pre-existing diabetes mellitus, neurological disorders, a history of urinary tract surgery, anatomical abnormalities of the urinary tract and active urinary tract infection. The investigations included a urinary symptom questionnaire with questions about urgency, stress and urge incontinence and then twin-channel subtracted cystometry was performed with a pressure-flow study at the end of the procedure. These investigations were performed in the third trimester & 12 weeks postpartum.

Results

39 women had complete pressure flow data before and after childbirth.

The average age was 29 years (range 18-39) and average gestational age at 1st visit – 36 weeks (range 34-39). Thirty women delivered vaginally (6 had an instrumental delivery) and 9 underwent a Caesarean section (3 pre-labour).

All women were continent prior to pregnancy. During pregnancy 17(46%) reported stress incontinence and postpartum 10(26%) reported stress incontinence.

The UOP and UCP before and after delivery are shown in Table One. There was a statistically significant reduction in UOP in women who delivered vaginally ($p<0.05$) compared to caesarean section but no significant changes in UCP.

There was no relationship between UOP &UCP in pregnancy & postpartum and the symptom of stress incontinence.

Table One -UOP and UCP before and delivery according to mode of delivery.

		Antenatal	Postnatal
Vaginal delivery	UOP	37 (19)	23 (27)*
(n=30)	UCP	30 (21)	24 (29)
Caesarean section	UOP	29 (29)	28 (19)
(n=9)	UCP	27 (31)	26 (22)

UOP = urethral opening pressure (cmH_20) UCP = urethral closure pressure (cmH_20)

*p=<0.05, All data presented as mean values (standard deviation).

Table 2. UOP and UCP before and after delivery in women reporting stress incontinence compared to asymptomatic women.

	Detrusor opening pressure*		Detrusor closure pressure*	
	Antenatal	Postnatal	Antenatal	Postnatal
Asymptomatic	35(19) (n=22)	41(14) (n=10)	30(23) (n=22)	46(17) (n=10)
Symptomatic	35 (25) (n=17)	33 (24) (n=29)	30(25) (n=17)	28(25) (n=29)

*cmH_2O All data presented as means (standard deviation)

Conclusions

This is the first study to demonstrate a reduction in urethral resistance after vaginal delivery compared to Caesarean section. This suggests that vaginal delivery damages the urethral sphincter mechanism.

The lack of relationship between urethral opening and closure pressures and symptoms may be because of the small size of this study, but more likely as the symptom of stress incontinence may reflect both underlying detrusor overactivity or urodynamic stress incontinence.

A larger study, with long-term follow-up is required to assess if these changes in urethral sphincter function are associated with the subsequent development of urinary incontinence. This again raises the question of promoting elective Caesarean section to protect the urinary continence mechanism.

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